A DISSERTATION ON

AETIOLOGICAL ANALYSIS OF OCULAR MOTOR NERVE (III , IV AND VI) PALSIES

M.S. DEGREE BRANCH (III)

OPHTHALMOLOGY

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

MARCH 2007

CERTIFICATE

This is to certify that this dissertation entitled "AETIOLOGICAL ANALYSIS OF OCULAR MOTOR NERVE (III, IV AND VI) PALSIES" submitted by DR.V. VIMALA to the faculty of Ophthalmology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement of the award of M.S.Degree Branch III (Ophthalmology) is a bonafide reserach work carried out by her under my direct supervision and guidance.

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DECLARATION

I, Dr. V. VIMALA solemnly declare that the dissertation titled "AETIOLOGICAL ANALYSIS OF OCULAR MOTOR NERVE (III , IV AND VI) PALSIES" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S., degree (Branch III Ophthalmology) Examination to be held in MARCH 2007.

Place : Madurai

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ACKNOWLEDGEMENT

I am grateful to The Dean, Madurai Medical College, Madurai for permitting me to do the study.

I am extremely grateful to Professor Dr. R. GeethaRamani. M.S. D.O., Professor and HOD of Ophthalmology, Madurai Medical College, Madurai for the able guidance, inspiration and encouragement she rendered at every stage of the study.

I take this opportunity to express my deep sense of gratitude to Professor Dr. R. Unnamalai M.S. D.O. for her guidance and help for executing my study.

I am grateful to Dr. T. Badrinarayanan M.S,D.O Asst. Professfor, Department of Ophthalmology for his valuable guidance, support and encouragement rendered to me during the study.

I am extremely grateful to all the Assistant professors, Department of Ophthalmology for having helped during the study.

I am extremely grateful to all the Assistant professors of Medicine and Neurology department for their support during the study.

I thank my study subjects who formed the back bone of the study and without whom this work would not have been possible.

Last but not the least, I thank “God, the Almighty” for being my guiding light all the way.

ABBREVIATIONS

AIDS	-	Acquired immunodeficiency syndrome
ARMD	-	Age related macular degeneration
BP	-	Blood pressure
BS	-	Blood sugar
COA	-	Consecutive optic atrophy
CSME	-	Clinically significant macular edema
CT	-	Computed tomography
DM	-	Diabetes mellitus
DR	-	Diabetic retinopathy
ENT	-	Ear, Nose and Throat
EOM	-	Extra ocular movements
ESO	-	Esotropia
EXO	-	Exotropia
F	-	Female
FBS	-	Fasting blood sugar
GTT	-	Glucose tolerance test
HbA1c	-	Glycosylated hemoglobin
HTR	-	Hypertensive retinopathy
HT	-	Hypertension HPT - Hyper tropia
III N	-	III nerve
IO	-	Inferior oblique
IR	-	Inferior rectus
IV N	-	IV nerve
LE	-	Left eye

LIO - Left inferior oblique

LIR - Left inferior rectus

LPS - Levator palpabrae superioris

LR - Lateral rectus

LSO - Left superior oblique

LSR - Left superior rectus

M - Male MR - Medial rectus

MR. Scan - Magnetic resonance scan

MRA - Magnetic resonance angiography

MRI - Magnetic resonance imaging

NPDR - Non proliferative diabetic retinopathy

NSAID - Non steroidal anti inflammatory drugs

OC Pills - Oral contraceptive pills

PDR - Proliferative diabetic retinopathy

PNS - Para nasal sinuses

PPBS - Post prandial blood sugar

RAPD - Relative afferent pupillary defect

RE - Right eye

RIO - Right inferior oblique

RIR - Right inferior rectus

RSO - Right superior oblique

RSR - Right superior rectus

SO - Superior oblique SR - Superior rectus

VDRL - Venereal disease research laboratory

VI N - VI nerve

CONTENTS

INTRODUCTION

A perfect alignment between the motor system of two eyes is responsible for viewing an object as single. The extraocular muscles of both eyes work in co-ordination. When any one or more of these falter , it may manifest as double vision , deviation of eyes or sometimes with pain. Patients may sometimes present to the ophthalmologist for one of these complaints ,may be referred by another physician or be seen accidentally while they just come for a routine checkup. This may be one of the first manifestation of a serious emergency like a posterior communicating artery aneurysm or a multisystem disease like diabetes. So every case has to be evaluated and its probable etiology identified. Here lies the role of an ophthalmologist to decide whether follow up and observation will suffice or may require emergency intervention by a neurosurgeon.

OCULOMOTOR NERVE

ANATOMY

The third cranial nerve is entirely motor in function. It supplies all the extraocular muscles of the eyeball except the lateral rectus and superior oblique. It also supplies the intraocular muscles namely the sphincter pupillae and the ciliary muscle.

FUNCTIONAL COMPONENTS

1. SOMATIC EFFERENT – concerned with movements of the eyeball.
2. GENERAL VISCERAL EFFERENT (parasympathetic)- for accommodation and contraction of the pupil.
3. GENERAL SOMATIC AFFERENT – for carrying proprioceptive impulses from the muscles supplied by the third nerve.

THE OCULOMOTOR NUCLEAR COMPLEX

LOCATION

In the midbrain at the level of superior colliculus in the ventromedial part of the central gray matter that surrounds the cerebral aqueduct.

It is a longitudinal column 10 mm long extending above from the floor of the third ventricle and below it is related to the nucleus of the fourth nerve. There are two motor nuclei:

1. main motor nucleus of large multipolar neurons

2. accessory Edinger westphal nucleus of small multipolar neurons.

The main motor nucleus has the following subnuclei:

1. DORSOLATERAL NUCLEUS- supplies ipsilateral inferior rectus
2. INTERMEDIAL NUCLEUS – supplies ipsilateral inferior oblique
3. VENTROMEDIAL NUCLEUS – supplies ipsilateral medial rectus
4. PARAMEDIAL NUCLEUS – supplies contralateral superior rectus

5.CAUDAL CENTRAL NUCLEUS – supplies bilateral levator palpebrae superioris.

The Edinger westphal nucleus lies posterior to the main oculomotor nuclear mass .It consists of a median and two lateral parts. It gives rise to preganglionic parasympathetic fibres.

CONNECTIONS OF THE NUCLEUS

1. Cerebral cortex

- motor cortex of both sides through the corticonuclear tracts.
- visual cortex through the superior colliculus.
- frontal eye field

2. Nuclei of 4 ,6 and 8 cranial nerves through the medial

longitudinal fasciculus.

3. Pretectal nucleus of both sides

4. Vertical and torsional gaze centres

5. Cerebellum through the vestibular nuclei.

COURSE AND DISTRIBUTION

It can be divided into four parts

1. the fascicular part
2. the basilar part
3. the intracavernous part
4. the intraorbital part

THE FASCICULAR PART

It consists of efferent fibres that pass from the third nerve nucleus through the red nucleus and the medial aspect of cerebral peduncle. They then emerge from the midbrain and pass into the interpeduncular space.

THE BASILAR PART

It consists of a series of 15 – 20 rootlets in the interpeduncular fossa. They coalesce to form a large medial and a small lateral root , which unite to form a flattened nerve , which then gets twisted bringing the inferior fibres superiorly and viceversa . Thus the nerve becomes a rounded cord . The nerve then passes between the posterior cerebral artery and the superior cerebellar artery . Then it runs

forward in the interpeduncular cistern (running lateral and parallel to the posterior communicating artery) to reach the cavernous sinus .

The intracavernous part :

The nerve enters the cavernous sinus by piercing the posterior part of its roof on the lateral side of the posterior clinoid process. It then descends on the lateral wall of the sinus , where it lies above the trochlear nerve . In the anterior part of the cavernous sinus , the nerve divides into superior and inferior divisions which enter the orbit through the middle part of the superior orbital fissure within the annulus of zinn.

The intraorbital part :

In the orbit the smaller superior division ascends on the lateral side of the optic nerve and supplies the superior rectus and levator palpebrae superioris . The larger inferior division divides into three branches :

i. Nerve to medial rectus passes inferior to optic nerve

ii. Nerve to inferior oblique passes (longest of the three branches) in between the inferior rectus and lateral rectus and supplies the oblique from its posterior border . It gives off the motor root to the ciliary ganglion .

iii. Nerve to inferior rectus passes and enters the muscle on its upper aspect.

The features of third nerve palsy : It may be complete or incomplete and it may be congenital or acquired.

1. Ptosis - due to paralysis of LPS
2. Deviation – eyeball is turned down , out and slightly intorted due to unopposed action of the lateral rectus and the superior oblique
3. Ocular movements – restriction of the following movements :

i. adduction - due to paralysis of medial rectus,

ii.elevation - due to paralysis of superior rectus and inferior oblique,

iii. depression - due to paralysis of inferior rectus and

iv. extorsion - due to paralysis of inferior rectus and inferior oblique.

4. Pupil - is fixed and dilated due to paralysis of sphincter pupillae
5. Accomodation - completely lost due to paralysis of ciliary muscle
6. Crossed diplopia – appears on manually raising the eyelid, which occurs due to paralytic divergent squint
7. Head posture - if the pupillary area is uncovered the head takes a posture consistent with the directions of actions of paralysed muscle i.e head is turned to the opposite side , tilted towards the same side and chin is slightly raised.

TOPOGRAPHIC LOCALISATION OF III NERVE PALSY

Nuclear III nerve palsy

- Extremely rare

- The arrangement of third nerve subnuclei requires the following criteria for diagnosis of nuclear lesion .

i. The superior rectus is innervated by the contralateral III nerve nucleus. Therefore , in a nuclear III nerve palsy there has to be paresis of contralateral superior rectus.

ii. Both levators are innervated by a single subnucleus – the caudal central nucleus . Therefore in a nuclear III nerve palsy there has to be bilateral ptosis.

III NERVE FASCICLE SYNDROME

Topical diagnosis depends upon the coexistence of other neurologic signs . Fascicles have already left the III nerve nucleus, so that the ocular manifestations are present on only one side.

Nothnagel syndrome

i. Lesion in the area of superior cerebellar peduncle.

ii. Ipsilateral III nerve palsy and cerebellar ataxia.

Benedikt syndrome

i. Lesion in the region of red nucleus.

ii. Ipsilateral III nerve palsy with contralateral hemitremor.

Weber syndrome

i. Involvement of the III nerve in the neighbourhood of the cerebral peduncle .

ii. Ipsilateral III nerve palsy with contralateral hemiparesis.

Claude syndrome

Features of both Benedikt and Nothnagel syndrome

UNCAL HERNIATION SYNDROME

In its course towards the cavernous sinus , the III nerve rests on the free edge of the tentorium cerebelli . The portion of the brain overlying the III nerve , at the tentorial edge , is the uncal portion of the under surface of the temporal lobe .

A supratentorial space occupying lesion , located anywhere in or above this cerebral hemisphere , may cause downward shift and herniation of the uncus across the tentorial edge , thereby compressing the III nerve.

A dilated and fixed pupil may be the first indication that altered consciousness may be due to a space occupying lesion .

POSTERIOR COMMUNICATING ARTERY ANEURYSM

In its course towards cavernous sinus , III nerve travels lateral to the posterior communicating artery.

The most common cause of nontraumatic III nerve palsy with pupillary involvement is an aneurysm at the junction of the posterior communicating artery and the internal carotid artery.

Hemorrhage suddenly enlarges the aneurysmal sac to which the III nerve is adherent or there may be actual hemorrhage into the substance of the nerve .

On occasion , the pupil is spared early in the course of aneurysmal compression of the III nerve .The patient must be followed carefully for the initial 5 -7 days to be certain about the status of the pupil.

CAVERNOUS SINUS SYNDROME

III nerve paresis is usually seen in association with other cranial nerve involvement ; IV ,V ,VI and oculosympathetic paralysis. There may be a partial involvement.

The pupil may be constricted due to coexisting horner syndrome or only minimally dilated due to both parasympathetic and sympathetic involvement .There may be aberrant regeneration of III nerve.

THE ORBITAL SYNDROME

Just before entering the superior orbital fissure , the III nerve splits into two divisions :

Superior division innervates – SR and LPS

Inferior division innervates - IR ,MR , IO , sphincter pupillae and ciliary muscle.

Therefore orbital involvement of the III nerve may result in selective paresis of structures innervated by only one of the divisions.

PUPIL SPARING ISOLATED III NERVE PARESIS

The pupillomotor fibres of the III nerve travel in the outer layers of the nerve and are therefore closer to the nutrient blood supply enveloping the nerve . The outer fibres are supplied by the pial plexus whereas the inner fibres are supplied by the vasa nervorum.

So this explains why the diabetics where the vasa nervorum are affected have pupillary sparing in 80 % and similarly in any ischemic vascular etiology . In contradiction when compressive lesions involve the III nerve the superficial fibres are affected resulting in pupillary involvement in 90 % .

Most patients with ischemic III nerve paresis demonstrate improvement in motility measurements within one month or may have complete recovery by 3 months (maximum : 6 months) .

Cranial imaging like MR scanning - MRI , MRA , Four vessel angiography and Lumbar puncture are recommended if :

i. The pupil is involved i.e dilates or becomes dilated in the initial 5 – 7 days after onset.
ii. No significant improvement in 3 months.
iii. The patient develops signs of aberrant regeneration of III nerve .
iv. Other neurologic findings develop .

DIAGNOSTIC GUIDELINES

The size and reactivity of the pupil are major determinants in patient evaluation . Other factors include :

1. Age of the patient.
2. Degree of somatic (motility , lid) involvement.
3. Some pupillary involvement is found in upto one – third patients with ischemic third nerve palsies.
4. Pupillary involvement may develop during the initial 5 – 7 days of a compressive third nerve palsy.
4. Periorbital pain does not distinguish between compressive or vasculopathic etiology .

EVALUATION GUIDELINES

1. All children less than 10 years should undergo MRI and MRA. If normal , proceed with cerebral angiography.
2. All patients older than 10 years with pupil involving third nerve palsy should undergo MRI and MRA .
3. All patients 10 – 50 years with pupil sparing III nerve palsy should undergo MRI and MRA . If normal then further steps are :
   i. Medical evaluation – eg .diabetes , hypertension.
   ii. Observation for pupillary involvement.
   iii. Followup for development of other neurologic abnormalities.
3. For all patients 50 years and older with pupil sparing III

nerve palsy with total somatic involvement :

i. Observation

ii. Medical workup – diabetes , hypertension and giant cell arteritis

5. All patients 50 years and older with partial pupillary involvement (anisocoria > 2 mm but pupil not fixed and dilated) should undergo MRI and MRA . If results are normal then cerebral angiography is considered .

INCIDENCE OF VARIOUS CAUSES OF III NERVE PALSY

1. Although neoplasm , aneurysm and ischemia are the most common etiologies , 10 – 25 % have an undetermined cause .

2. Approximately one half of III nerve palsies in children are congenital and a high percentage have signs of aberrant regeneration. About 10 - 20 % are due to aneurysm or neoplasm. Therefore all children should undergo MR scanning.

ABERRANT REGENERATION OF III NERVE

Regeneration of aberrant III nerve fibres may result in fibres meant for one structure being hooked up (axon sprouting) with fibres that terminate in another structure.

Clinical phenomena :

Lid gaze dyskinesis :

- Inferior rectus may end up innervating the levator so that the lid retracts when the patient looks down - PseudoVon Graefe Sign.
- Some of the medial rectus fibres end up supplying some of the innervation to the levator so that the lid retracts when the patient adducts the eye - Inverse Duane syndrome.

Pupil gaze dyskinesis :

Medial rectus fibres may end up innervating the pupillary sphincter muscle so that pupil constricts better during convergence and adduction than as a response to light – Pseudo Argyll Robertson pupil .In true Argyll Robertson pupil , the pupil constricts only during convergence and not during adduction. Some inferior rectus fibres may be supplying pupillary sphincter , so that on attempted downgaze , the pupil constricts.

TWO FORMS OF ABERRANT REGENERATION

1 . Primary aberrant regeneration

- No preceding acute III nerve palsy.
- Insidious development of III nerve palsy with signs of misdirection .
- Sign of intracavernous lesion , meningioma ,aneurysm or neurinoma.

2. secondary aberrant regeneration

Observed weeks to months after acute III nerve palsy during recovery.

Seen after trauma and tumor compression of the III nerve , but never after an ischemic III nerve paresis .If patient is followed with a presumed diagnosis of ischemic III nerve palsy and then develops signs of aberrant regeneration , then MR scanning and cerebral angiography are indicated.

RARE CAUSES OF III NERVE PALSY

1 . Minor head trauma :

Usually head trauma that causes III nerve palsy is severe enough to cause loss of consciousness and other neurologic deficits.

A patient who harbours a basal intracranial tumor can develop III nerve palsy with only minor head trauma . So a minimal head injury resulting in III nerve palsy is an indication for cranial MR scanning.

2 . Opthalmoplegic migraine :

- Onset always in childhood .
- Family history usually present.
- III nerve palsy can occur anytime in relation to headache but usually appears as the headache phase abates .
- Usually III nerve palsy clears completely in one month, but occasionally permanent oculomotor paresis occurs.

3. Cyclic oculomotor palsy :

- Usually present at birth or in early childhood .
- Occurs in the setting of a total III nerve palsy .
- Spastic movements of the muscles innervated by III nerve results in lid elevation , adduction , miosis and increased accommodation.
- These movements occur at regular intervals lasting 10 – 30 seconds.
- Etiology unknown .

TROCHLEAR NERVE

The trochlear nerve is entirely motor in function and supplies only the superior oblique muscle of the eyeball.

PECULIARITIES

- The only cranial nerve to arise from the dorsal aspect of the brain .
- The only cranial nerve to cross completely to the other side i. e. the trochlear nerve arises from the contralateral nucleus .
- The longest and thinnest of all cranial nerves.

FUNCTIONAL COMPONENTS

1 . SOMATIC EFFERENT – concerned with the primary,secondary and tertiary actions of superior oblique .

2 . GENERAL SOMATIC AFFERENT – carries proprioceptive impulses from the superior oblique . The impulses are relayed to the mesencephalic nucleus of the trigeminal nerve.

NUCLEUS

Situated in the ventromedial part of the central gray matter of the midbrain at the level of inferior colliculus . It is caudal to and continious with the III nerve nuclear complex . It belongs to the somatic efferent column of nuclei .

CONNECTIONS OF THE NUCLEUS

1 . Cerebral cortex

i. Motor cortex – of both sides through the corticonuclear tracts .

ii. Visual cortex - through the superior colliculus

iii . Frontal eye fields .

2. Nuclei of 3, 6 and 8 cranial nerves through the medial longitudinal bundle .

3. Superior colliculi through the descending predorsal bundle.

4 . Vertical and torsional gaze centres.

5 . Cerebellum through the vestibular nuclei.

COURSE AND DISTRIBUTION

It is divided into

i) the fascicular part
ii) the precavernous part
iii) the intracavernous part
iv) the intraorbital part .

THE FASCICULAR PART

It consists of efferent fibres which after leaving the nucleus , pass posteriorly around the aqueduct in the central gray matter and decussate completely in the anterior medullary velum.

THE PRECAVERNOUS PART

The trochlear nerve trunk emerges from the superior medullary velum just below the inferior colliculus on the dorsal aspect of midbrain.

It then winds round the superior cerebellar peduncle and the cerebral peduncle just above the pons. It runs beneath the free edge of the tentorium , and like the III nerve passes between the posterior cerebral and superior cerebellar arteries to appear ventrally lateral to cerebral peduncle . It then pierces the dura on the posterior corner of the roof of the cavernous sinus to enter into it .

THE INTRACAVERNOUS PART

In the cavernous sinus, the nerve runs forwards in its lateral wall lying below the III nerve and above the first division of the fifth cranial nerve . In the anterior part of the cavernous sinus, it rises, crosses over the III nerve and leaves the sinus to pass through the lateral part of the superior orbital fissure (where it passes superolateral to annulus of zinn and medial to frontal nerve).

THE INTRAORBITAL PART

After entering through the lateral part of the superior orbital fissure, the nerve passes medially above the origin of the LPS and ends by supplying the superior oblique on its orbital surface.

The number of fibres in the intraorbital part of the trochlear nerve are greater than its intracranial part . These extra fibres carrying the proprioceptive impulses from the superior oblique leave the trochlear nerve to join the ophthalmic division of fifth nerve in the cavernous sinus.

FEATURES OF IV NERVE PALSY

1. Hyperdeviation - due to weakness of superior oblique. This becomes more obvious when the head is tilted towards ipsilateral shoulder (Park Bielchowsky head tilt test).
2. Ocular movements - depression is limited in adduction . Intorsion is also limited.
3. Diplopia – vertical diplopia occurs on looking down
4. Abnormal head posture – To avoid diplopia head adopts a posture such that the action of superior oblique is less needed .i .e .face is slightly turned to opposite side , chin is depressed and head is tilted towards the opposite side .

PARKS BIELCHOWSKY THREE STEP TEST

1. The medial and lateral rectus muscles do not have a vertical action. Therefore hypertropia of paretic etiology is due to weakness of one or more of the following vertically acting muscles

   a. RIO ; LIO b. RSO ; LSO c. RIR ; LIR d. RSR ; LSR

2. If the HPT is due to weakness of only one of these eight muscles, answering the following three questions identifies the paretic muscle.

   Each step cuts the possible number of muscles into half .

3. First step – which is the higher eye ?

a) If the patient has a RHPT then the weak muscle is either a depressor of the RE (RIR / RSO) or an levator of the LE(LSR / LIO).

b) If the patient has LHPT , then weak muscle is either an elevator of the RE (RSR / RIO) or depressor of the LE (LIR /LSO)

4 . Second step – HPT worse on right or left gaze?

- The vertical rectus muscles (superior and inferior recti) have their greatest vertical action (and least torsional action) when the eye is abducted . The oblique muscles (superior and inferior obliques) have their greatest vertical action (and least torsional action) when the eye is adducted .

So in each case,

i. RHPT worse on gaze right (RIR / LIO)

ii. RHPT worse on gaze left (RSO / LSR)

iii. LHPT worse on gaze right (LSO / RSR)

iv. LHPT worse on gaze left (RIO / LIR)

5. Third step – Is the HPT worse on head tilt right or left ?

a. The superior muscles (SR and SO) intort the eye ; the inferior muscles (IR and IO) extort the eye.

b. When the head is tilted to the right , right eye will be intorted by the contraction of the RSR and RSO ; these two muscles work together in effecting the intorsion and neutralize each others vertical

action (RSR is an elevator and RSO is a depressor) .

c. If one of these muscles is the paretic muscle responsible for the HT, then the vertical action will not be neutralized and the HT will be worse on tilting the head to the right shoulder .

CLINICAL SYNDROMES

1. NUCLEAR FASCICULAR SYNDROME

To differentiate nuclear from fascicular lesions is impossible due to the short course of the fascicles within the midbrain , thus there are no associated neurologic signs.

Common causes : hemorrhage , infarction , demyelination and trauma. Fascicular lesion may be seen with contralateral horner syndrome, since the sympathetic pathways descend through the dorsolateral tegmentum of the midbrain adjacent to the trochlear fascicles.

2 . SUBARACHNOID SPACE SYNDROME

Fourth nerve is particularly susceptible to injury as it emerges from the dorsal surface of brainstem . When bilateral IV nerve palsies occur , the site of injury is likely in the anterior medullary velum. Contrecoup forces transmitted to the brainstem by the free tentorial edge may injure the nerves at this site.

Other causes : pinealoma , tentorial meningioma , meningitis and neurosurgical trauma.

3 . CAVERNOUS SINUS SYNDROME

There are associated other cranial nerve palsies - III , V ,VI and oculosymphathetic paralysis.

4 . ORBITAL SYNDROME

Associated with III , V and VI cranial nerve palsies and orbital signs – proptosis , chemosis and conjunctival injection.

Causes – trauma , inflammation and tumour .

5 . ISOLATED IV NERVE PALSY

i) Congenital -

Most often seen in pediatric population and late in life (V – VII) decades as patient IV nerve palsy may decompensate.

Diagnosed by

- Large vertical fusion amplitude.
- Family photograph – old photos may detect long standing tilt and indicate congenital etiology.

ii) Acquired –

- Acute onset of vertical diplopia , usually with torsional component .

– As with other isolated ocular motor neuropathies , if the IV nerve palsy has not improved or recovered within four months or if neurologic signs develop , further workup is indicated .

MEASURING THE TORSIONAL COMPONENT OF IV NERVE PALSY

Double Maddox rod test is used to quantitate torsional component of diplopia . The patient complains of intorsion of the image seen by the eye with IV nerve palsy . This indicates extorsion of the patient eye caused by overaction of the antagonist inferior oblique muscle . Greater than 10 degrees torsion is suggestive of bilateral IV nerve palsies.

Many adults presenting in the V and VI decades may have decompensated congenital IV nerve palsy .

DIFFERENTIAL DIAGNOSIS OF VERTICAL DIPLOPIA

1. Ocular myasthenia
2. Thyroid eye disease
3. Orbital disease (tumour , trauma , inflammation , blow out fracture etc.)
4. III nerve palsy
5. Brown syndrome
6. Skew deviation

SYNDROMES OF SUPERIOR OBLIQUE

1. Brown (sheath) syndrome
   - There is limitation of elevation of the eye in adduction

because movements of the superior oblique tendon in the trochlea are restricted whereas elevation in abduction is normal.

- Affected eyes are usually hypotropic ; patient develops an abnormal head posture (chin up) .
- Forced duction test is positive.
- In the congenital type superior oblique tendon is short and tethered. The acquired type may be due to tenosynovitis or trauma to the trochlear region .

2 . Superior oblique myokymia

- Unexplained vertical diplopia
- Paroxysmal, rapid, vertical and torsional movements of one eye.
- Usually benign , occasionally seen with multiple sclerosis or posterior fossa tumour .
- Treated with carbamazepine , propranolol and gabapentin .
- Superior oblique surgery is also considered if not responding.

ABDUCENT NERVE

It is an entirely motor nerve that supplies the lateral rectus muscle of the eyeball .

FUNCTIONAL COMPONENTS

i. SOMATIC EFFERENT - for lateral movements of the eye .

ii. GENERAL SOMATIC AFFERENT - for proprioceptive impulses from the lateral rectus muscle . These impulses ultimately reach the mesencephalic nucleus of the trigeminal nerve .

NUCLEUS

Situated in the lower part of pons , close to the midline beneath the floor of the IV ventricle . It is closely related to the fasciculus of the facial nerve . It consists of two types of multipolar cells - large and small. The large multipolar cells give rise to fibres of the abducent nerve , while the fibres of the small multipolar cells relay in the oculomotor nucleus via the medial longitudinal fasciculus . The small multipolar cells are believed to form the paraabducent nucleus. Since the abducent nucleus belongs to the group of somatic efferent nuclei, it lies in line with the nuclei of IV and III nerves above and the hypoglossal nucleus below.

CONNECTIONS OF THE NUCLEUS

1 . Cerebral cortex –

i. Motor cortex (precentral gyrus) through the afferent corticonuclear fibres from both cerebral hemispheres.

ii) Visual cortex , through the superior colliculus.

iii) Frontal eye fields

2. Nuclei of III , IV and VIII cranial nerves through the medial longitudinal bundle.

3. Pretectal nucleus of both sides .

4. Horizontal gaze centre through the medial longitudinal bundle.

5. Cerebellum through vestibular nuclei.

COURSE AND DISTRIBUTION

It is divided into i. the fascicular part

ii. the basilar part

iii. the intracavernous part and

iv. the intraorbital part .

THE FASCICULAR PART

It consists of efferent fibres which start from the nucleus, pass forward traversing the medial lemniscus and pyramidal tract. These then emerge by 7 – 8 rootlets from the junction of pons and medulla just lateral to the pyramidal prominence of medulla . The rootlets join to form one nerve , at varying distances from the origin.

THE BASILAR PART

The nerve then runs forwards , upwards and slightly laterally through the cisterna pontis between the pons and clivus. The nerve then runs upwards on the back of petrous temporal bone near its apex . At the sharp upper border of the petrous bone, the nerve bends forward at right angles under petrosphenoidal ligament through the Dorello's canal and enters the cavernous sinus by piercing its posterior wall at a point lateral to the dorsum sellae and superior to the apex of petrous temporal bone .

THE INTRACAVERNOUS PART

In the cavernous sinus , the nerve runs horizontally forward, occupying a position below and lateral to the internal carotid artery. The internal carotid artery is surrounded by symphathetic plexus . The nerve then leaves the cavernous sinus to enter the orbit through the middle part of the superior orbital fissure through the annulus of zinn. In the superior orbital fissure, the abducent nerve lies inferolateral to the oculomotor and nasociliary nerves .

THE INTRAORBITAL PART

In the orbit the nerve runs forwards and enters the ocular surface of the lateral rectus muscle just behind its middle portion after dividing into three or four branches .

CLINICAL FEATURES OF VI NERVE PALSY

1. Deviation - In the primary position , the eyeball is convergent due to unopposed action of the medial rectus muscle .
2. Ocular movements - Abduction is restricted .
3. Diplopia – Uncrossed horizontal diplopia occurs , worse towards the action of paralysed muscle .

4 . Head posture – The face is turned towards the action of paralysed muscle to minimize diplopia .

CLINICAL SYNDROMES

1.THE BRAINSTEM SYNDROME

A brainstem lesion of the VI nerve also affects V, VII ,VIII nerves and the cerebellum . The sixth nerve nucleus contains otorneurons that supply the lateral rectus muscle and abducens internuclear neurons that project via the medial longitudinal bundle to the medial rectus subdivision of the contralateral oculomotor nucleus. Thus a nuclear VI nerve palsy causes an ipsilateral conjugate gaze palsy .

When other adjacent structures are affected , there are associated findings like paralysis of

i. Oculosympathetic central neuron – ipsilateral horner syndrome
ii . Pontine paramedian reticular formation - ipsilateral horizontal conjugate gaze palsy.
iii. Medial longitudinal fasciculus - ipsilateral internuclear ophthalmoplegia .

iv. Pyramidal tract – contralateral hemiparesis .

So the brainstem syndrome may consist of any combination of the deficits listed above . Of these the common ones are :

i. Millard –Gubler syndrome

- VI nerve paresis
- Ipsilateral VII nerve paresis
- Contralateral hemiparesis

ii. Raymond – Cestan syndrome

- VI nerve paresis
- Contralateral hemiparesis

iii. Foville syndrome

- Horizontal conjugate gaze palsy
- Ipsilateral V , VII and VIII cranial nerves
- Ipsilateral horner syndrome

2 .THE SUBARACHNOID SPACE SYNDROME

A rise in intracranial pressure results in downward displacement of the brainstem , with stretching of the VI nerve which is tethered at its exit from the pons and in Dorello canal.

- It gives rise to nonlocalising VI nerve palsies of raised intracranial pressure .
- 30 % patients with pseudotumour cerebri have VI nerve paresis , associated with papilledema and its visual field defects. Otherwise

disturbances in the subarachnoid space causing VI nerve palsies include hemorrhage , meningeal or parameningeal infection (e.g . viral, bacterial, fungal), inflammation (e.g. sarcoidosis) or infiltration (e.g. lymphoma, leukemia and carcinoma).

THE PETROUS APEX SYNDROME

The VI nerve within Dorello's canal is susceptible to pathologic processes affecting the petrous bone .

i .Gradenigo syndrome

- It is due to localized inflammation or extradural abcess of petrous apex following complicated otitis media .
- VI nerve palsy
- Ipsilateral hearing deficit
- Ipsilateral facial pain in the distribution of V nerve .
- Ipsilateral facial paralysis

ii. Petrous bone fracture

- Basal skull fracture following head trauma.
- Potential cranial nerve involvement – V , VI ,VII and VIII
- Associated findings – hemotymphanum, Battle sign (mastoid ecchymosis) , cerebrospinal fluid otorrhoea etc .

3. Pseudo Gradenigo syndrome

- Nasopharyngeal carcinoma may cause serous otitis media due to obstruction of the eustachian tube and the carcinoma may subsequently invade the cavernous sinus , causing VI nerve paresis.

- Cerebellopontine angle tumour may cause VI nerve paresis, decreased hearing , V and VII nerve palsy with ataxia and papilledema .

THE CAVERNOUS SINUS SYNDROME

The associated findings are

- III , IV and ophthalmic nerve involvement
- Horner syndrome
- May be associated with optic nerve / chiasmal / pituitary disease . Causes – trauma , vascular , neoplastic and inflammatory .

THE ORBITAL SYNDROME

- Proptosis , chemosis and conjunctival injection.
- The optic nerve may appear normal or demonstrate atrophy or edema .
- Trigeminal signs are limited to the ophthalmic division.
- It becomes frequently difficult to distinguish between cranial nerve palsy and mechanical restriction of the globe .
- Causes – tumour , trauma , inflammatory pseudotumour and cellulitis.

ISOLATED VI NERVE PALSY

Frequently seen as a postviral syndrome in young patients or as a ischemic mononeuropathy in adults . If an ocular motor nerve palsy occurs in a young patient there is a possibility of a neoplasm so aggressive workup must be considered . If an ocular motor nerve palsy occurs in a older patient there is a greater likelihood of an ischemic mononeuropathy a less aggressive workup is needed .

RADIOGRAPHIC STUDIES

- Patients under age 40 should undergo cranial MR scanning .
- Patients less than 15 years may have a postviral etiology .
- If patients older than 40 present with VI nerve palsy, the most likely cause is an ischemic mononeuropathy. If it doesn't resolve in 4 months or if other cranial nerve involvement occurs, then evaluation is needed like :
    - Medical and neurologic examination
    - CT scan
    - MRI
    - Lumbar puncture
    - Cerebral angiography

ETIOLOGY

- 8 – 30 % is undetermined.
- 16 – 30 % is miscellaneous (leukemia, migraine, pseudotumour cerebri, multiple sclerosis and myelography). This miscellaneous group reflects the poor localizing value of palsies of the VI nerve .

DIFFERENTIAL DIAGNOSIS

1. Thyroid eye disease
2. Myasthenia gravis
3. Duane syndrome
4. Spasm of the near reflex
5. Medial wall orbital blow out fracture
6. Break in fusion of a congenital esophoria

CAUSES OF OCULOMOTOR PALSIES

1. Nuclear

- Infarction
- Demyelination
- Metastatic tumour

2. Fascicular

- Infarction
- Demyelination (rare)
- Tumour

3. Interpeduncular

- Aneurysm
- Trauma
- Meningitis

4. Cavernous sinus

- Carotid cavernous fistula
- Granulomatous inflammation (Tolosa Hunt syndrome)
- Intracavernous aneurysm
- Extrasellar extension of pituitary tumour
- Meningioma

- Sphenoid sinus carcinoma

- Metastatic tumour

- Mucormycosis (and other fungi)

- Herpes zoster

5 . Orbit

- Nonspecific inflammation (pseudotumour)

- Trauma

- Tumour

6 . Ischemic ophthalmoplegia (e.g diabetes ,hypertension etc.)

7 . Miscellaneous

- Polyneuritis (Guillian Barre Fisher syndrome)

- Cyclic oculomotor palsy (Bielchowsky)

- Migraine

- Arteritis

CAUSES OF PAINFUL OPHTHALMOPLEGIA

1 . Orbit

- Inflammatory pseudotumour
- Contiguous sinusitis
- Mucormycosis or other fungal infections
- Metastatic tumour
- Lymphoma

2 . Superior orbital fissure / Anterior cavernous sinus

- Nonspecific granulomatous inflammation (Tolosa Hunt syndrome)
- Metastatic tumour
- Nasopharyngeal carcinoma
- Lymphoma
- Herpes zoster
- Carotid cavernous fistula
- Cavernous sinus thrombosis

3 . Parasellar area

- Pitutary adenoma
- Intracavernous aneurysm
- Metastatic tumour

- Nasopharyngeal carcinoma
- Sphenoid sinus mucocoele
- Meningioma , Chordoma
- Petrositis

4 . Posterior fossa

- Posterior communicating artery aneurysm
- Basilar artery aneurysm (rare)

5 . Miscellaneous

- Diabetic ophthalmoplegia
- Migrainous ophthalmoplegia and Cranial neuritis

TREATMENT OF OCULAR MOTOR NERVE PALSIES

Follow up of cases of ocular motor nerve palsy that do not need urgent management , like the posterior communicating artery aneurysm must be at 6 weekly intervals till 6 months or two consecutive 6 weekly follow-ups reveal no change in motility. Every time diplopia charting , Hess charting , recording of deviations in nine gazes is done. During the meantime , patient is greatly disturbed by diplopia . So some nonsurgical modalities are practiced for symptomatic relief . If no resolution occurs after about 8 – 12 months then surgery is considered.

1. Prisms–are helpful in providing binocular vision as well as reducing the chances of development of contracture, but are useful only in small angle squints. Fresnel prisms are also used.
2. Botulinum toxin – the ipsilateral antagonist is paralysed by chemodenervation . The effect lasts for about 2 – 3 months . If necessary the injection can be repeated.
3. Occlusive prisms or opaque contact lens
4. Surgery – mainly to weaken the antagonist , usually ipsilateral and sometimes also the contralateral antagonist , in addition to strengthening the paralysed muscle . The amount of recession resection varies depending upon which eye habitually fixates

(secondary deviation or primary deviation needs to be corrected). Another principle is to restrain the contralateral antagonist by performing retroequatorial myopexy.

In the case of III nerve , the aim is to achieve diplopia free ocular position in primary position and downgaze .The latter should never be compromised for the upgaze .Anyway it is difficult because the III nerve supplies most of the extraocular muscles except two . Moreover aberrant regenerations alter the clinical picture . Each case has to be considered on a individual basis .

In the case of IV nerve , either strengthening of superior oblique or weakening ipsilateral inferior oblique or contralateral inferior rectus is done .The results of surgery for both congenital and acquired IV nerve palsy is excellent .

AIMS OF THE STUDY

1. To study the disease patterns, etiologies and pathogenesis of the infranuclear lesions of III, IV and VI cranial nerves (the ocular motor nerves)
2. To study the relationship of glycemic and hypertensive control in ischemic ocular motor nerve palsies.
3. To compare the usefulness of the various investigative techniques
4. To study the recovery pattern of the nerve palsies

MATERIALS & METHODS

The cases studied included those patients with neurogenic ocular motor nerve palsies who present to the ophthalmology department, Government Rajaji Hospital, Madurai as well as those who were referred from other departments like diabetology, neuromedicine, neurosurgery or the surrounding primary and secondary health care centres. All age groups and both sexes were included. A complete ophthalmological workup was done.

Inclusion Criteria :

1. All infranuclear ocular motor nerve palsies
2. III, IV, VI cranial nerves alone or in combination.

Exclusion Criteria :

All supranuclear, nuclear nerve palsies, myogenic and restrictive neuropathies

Registration :

Name
Age
Sex
Occupation
Address

History of present illness :

The common complaints were :

a. Double vision - whether uniocular / binocular, constant / intermittent, fluctuating or not, more for near or distance, whethere images are horizontally or vertically separated, whether it is increased on any particular direction, onset and progress.

b. Pain - headache / periorbital pain, location, nature, any radiation, aggravating and relieving factors, any association with nausea / vomiting.

c. Drooping of lids – unilateral / bilateral, total / partial

d. Defective vision – apart from double vision, any blurring or inability to see due to drooping of lid

e. Deviation of eyeball - right / left eye, duration

f. Abnormal head posture

g. Vertigo (sensation of rotation of self / surroundings)

Details of trauma, if any is recorded.

Details of the progress from onset, the treatment undergone to the present state is noted. Any other significant medical / surgical history is also recorded.

Past H/o

H/o diabetes, hypertension, tuberculosis, syphilis, AIDS, malignancy in the present or past.

H/o migraine or neurologic disease

H/o exanthems and vaccination

Personal H/o

Diabetes , smoking , alcoholism etc.

General Examination

General vital data like pulse, blood pressure, peripheral pulses are noted. Also gives an idea of the health status of the patient.

Ocular Examination :

- ✓ Head posture, facial symmetry are noted.
- ✓ Any deviation of eyeball is recorded. Under slit lamp, details of the anterior segment from the lids to the lens are noted.
- ✓ Extraocular movements are noted down – both ductions and versions. While looking for EOM, the aberrant innervation patterns are also looked for.
- ✓ Pupil size, reaction, any anisoconia is noted.
- ✓ A dilated fundus examination and refraction is done. Ptosis and proptosis if present are evaluated.
- ✓ Diplopia charting - is done in a dark room. Patient is asked to wear goggles with red in front of the right eye and green before the left eye. A torch light with a staenopic slit is used. The patient is asked to look at this torch held 120 cm away and then the torch is moved to various positions. The patient is asked to describe the position of the images. The false image is usually the fainter and farther one. Any tilt of the image is usually the fainter and farther one. Any tilt of the image and

variation in the distance between images at various positions is asked for.

- ✓ If a superior oblique palsy is suspected, Parks Bielchowsky's 3 step head tilt test is done.
- ✓ A forced duction test is performed in doubtful cases to rule out restrictive etiology.
- ✓ Tensilon test is performed in some cases to rule out myasthenia gravis.

Neurologic Examination :

Examination of other cranial nerves

Motor, sensory, cerebellar symptoms and signs.

Examination Of Thyroid :

Any neck swelling is looked for

Examination Of spine & back :

To look for congenital anomalies and neurocutaneous markers

Examination Of ENT structures

Investigations :

Both right and left eye (for all cases)

1. Vision - a. Uncorrected (Using Snellen's charts at 6 metres)
   b. Best corrected
2. Intraocular pressure - with schiotz tonometer after topial anaesthesia

3. Detailed slit lamp examination

   Lid

   Conjunctiva

   Cornea

   Iris

   Pupil

   Anterior Chamber

   Lens / Pseudophakia / Aphakia

4. Fundus examination - any abnormalities , diabetic retinopathy etc

5. Diplopia charting

6. Park 3 step test

7. Measurement of deviation - primary & secondary deviation, cover – uncover test in various gaze positions, for near and distance as well.

8. Hess charting

9 . Exophthalmometry

10. Visual field examination

Blood Tests : (for all cases)

Total count

Differential count

Erythrocyle sedimentation rate

Blood sugar - Fasting

Postprandial

In doubtful cases, Glucose tolerance test/HbA1c

Mantoux intradermal test

Serum cholesterol

Blood VDRL

Rheumatoid factor

Radiology (in indicated cases)

X ray orbit - fractures / erosion

X ray skull

X ray chest - tuberculosis

X ray PNS (paranasal sinuses) - mucococle, antral growth, sinusitis, orbit floor fractures

Orbital USG : (in indicated cases)

Neuro imaging : (in indicated cases)

CT : Sellar, parasellar tumours, anerusyms (intraluminal thrombosis and calcification), tumours of cerebellopontive angle, nasopharyngeal tumours with intracranial extension, basal skull fracture, hematoma etc.

MRI

MRA

Cerebral angiography

Specialist opinion (in indicated cases)

Diabetology

Otorhinolaryngologist

Neurophysician / Neurosurgeon

Radiologist

Follow up :

Recording of patient's complaints - whether stable / improving / worsening

- Vision
- Pupil assessment
- Extraocular movements
- Diplopia charting
- Fundus
- Examining for signs of pupillary involvement or abevant regeneration in cases of III N palsy
- Investigation

    Blood sugar FBS

    PPBS

    GTT / Hb A1c

    BP

    Imaging studies, if necessary

LITERATURE REVIEW

1. **Isolated and combined pareses of cranial nerves III, IV and VI**

A retrospective study of 412 patients[13]

Berlit P - Manheim Neurological clinic, University of Heidelberg

It was a retrospective study based on the medical records of 412 patients. Palsies of the III nerve (n=172) and VI nerve (n=165) were more frequent than IV nerve (n=25). Combined nerve palsies (n=50) were generally combinations of the III and IV (n=21) or paresis of all 3 cranial nerves (n=17). 165 ocular nerve palsies were due to vascular causes – in 135 of them diabetes and or hypertension was present. In inflammatory diseases and brain tumors the abducens nerve was the most frequently affected. The origin of ophthalmoplegia was unclear in 73 patients.

Ocular nerve paralysis was most common with tumours, aneurysms and vascular causes and in 206 cases was only partial. Pain was associated with tumour, trauma and aneurysm. In IV nerve palsies pain was much less frequent than in palsies of the other 2 ocular nerves. The most favourable prognosis was with inflammatory and vascular lesions. In the latter the outcome improved by the administration of NSAID.

J Neurol Sci. 1991 May, 103 (1), 10-5

2 . **Acquired palsy of the oculomotor, trochlear and abducent nerves**.

Tiffin PA, MaeEwen PJ, Craig EA, Clayton G

Dept. of Oph., Ninewells hospital, A medical school, Dundee, UK[2].

Eye 1996 PMID 8796166

A retrospective study was conducted on 165 patients between 1984 to 1992 in those with acquired III, IV and VI nerve palsies. The incidence of VI nerve palsy (57%), IV nerve palsy (21%). 111 nerve palsy (17%) and multiple nerve palsies (5%), Etiology was unknown in 35%, vascular in 32%, neoplastic 2% and aneurysm 1%.

Total recovery occurred (within 3 months) in 57% and atleast a partial recovery was seen in 80%.

3. **Risk factors for ischemic ocular motor nerve palsies** Jacobson DM, McCanna TD, Layde PM - Winconsin Arch ophthalmol 1994 Jul, 112 (7) ; 961 - 6

A case control study was conducted in 65 cases of ocular motor nerve palsies in patients 50 years and older hypercholesterolemia, coronary artery disease, LVH(left ventricular hypertrophy), adiposity, tobacco use, prior ocular motor nerve palsy and elevated hematocrit. In addition to the usual risk factors, LVH and elevated hematocrit were found to be important determinants.

4. Multiple cranial nerve palsies, analysis of 979 cases.

Keane JR. Arch Neurol 2005 Nov. ; 62 (11) : 1714 – 7

Department of neurology, Los Angeles, USA[19]

The study was done to identify the causes of multiple cranial nerve palsies. It was conducted in 979 patients with simultaneous or serial involvement of 2 or more different cranial nerves. The involvement of VI nerve (565), VII nerve (n=466), V nerve (353), III nerve (n=339) was documented . The locations and causes were diverse. The location was in cavernous sinus (n=252), brainstem (n=217) and individual nerves (n=182). The causes were tumours (n=305), trauma (n=128), vascular (n=128), infection (n=102) and Guillian Barre and Fisher syndrome (n=91). Recurrent cranial neuropathies were uncommon. (n=43) and with diabetes (n=14), self limited unknown (n=14) and idiopathic cavernous sinusitis (n=10). While the locations and causes of multiple cranial neuropathies are highly diverse, the fact that tumors comprise more than a quarter of cases places a premium on prompt diagnosis.

5. Longterm prognosis in patients with vasculopathic VI nerve palsy.

AmJ Ophthalmol 2002 Jul : 134 (1) : 81-4.

The study was conducted to determine longterm prognosis in patients with vasculopathic VI nerve palsy – specifically regarding the degree of recovery and incidence of recurrent similar episodes. 59 cases were

evaluated and the mean age group was 65.3 + 11.6 years. 51(86%) had complete resolution, 8 (14%) has incomplete resolution. Resolution was seen in 31%. The average number of recurrence was 1-4.

6. Curr Opin ophthalmol 1997 Dec : 8(6) : 45-57

University of Milano, Italy. Bianchi – Marzoli S, Brancato R

Topical diagnosis of III, IV and VI is required before imaging studies and the workup was performed. MRI is conformed to be the most useful tool for diagnosis is most cases. Finally, recently developed MRI techniques were found to be more sensitive than conventional MRI in severe cases.

7 .Extraocular motor dysfunction associated with tumours. Eskridge JB, School of optometry, Medical centre, Birmingham[12]

Optom clin 1993:3(3) : 135-160

Intracranial and extracranial tumors can be the cause of extraocular muscle dysfunction. In approximately 19%, paralysis of 3,4 and 6 nerves is due to neoplasm. Tumors cause extraocular muscle dysfunction by directly affecting extraocular muscles, directly affecting 3,4,6 cranial nerves or indirectly affecting the supranuclear areas, extraocular muscle areas, indirectly affecting any of the above by metastasis from a tumor in another part of the body and indirectly affecting 3,4,6 cranial nerves or their nuclei or the supranuclei or extraocular movement areas by raised intracranial pressure. The ocular symptoms and signs vary based on the mechanisms involved.

RESULTS

50 cases of ocular motor nerve palsies were examined. A prospective study was conducted .

1. AGE DISTRIBUTION

The following table shows the age distribution in the various types of nerve palsy.

Table – 1

AGE DISTRIBUTION IN OCULAR MOTOR NERVE PALSIES

Age Group	III N	IV N	VI N	Multiple Nerves	Total
1 – 15 yrs	1	-	2	-	2
15 – 30 yrs	2	3	1	-	7
30 – 45 yrs	4	3	6	1	14
45 – 60 yrs	6	1	10	2	19
> 60 yrs	3	-	4	1	8
Total	16	7	23	4	50

In the study of 50 cases, the cases with VI nerve palsy was maximal (23 cases – 46%), followed by the III nerve (16 cases – 32%). Next in frequency was IV nerve palsy (7 cases – 14%) and the least in frequency were multiple cranial nerve palsies (4 cases – 8%).

Regarding age distribution, considering all the nerve palsies in total, the maximum number of patients belonged to the 45-60 years group, followed in frequency by the 30-45 year group (14 cases). The least number was seen in 1-15 year group (only 3 cases).

Considering each nerve palsy, with regard to the III nerve, the maximum number was in 45-60 years group (6 cases of 16 – 37.5%). This signifies the high incidence of microangiopathic lesions due to vascular causes in this group. The incidence was less in the 1-15 year and > 60 year (extremes of age groups).

With regard to IV nerve, the total no. of cases being 7, the incidence was equal in 15-30 years and 30-45 years age group. (3 cases each – 42.86 % each respectively). This age group comprising the active outdoor population, are more prone to injuries especially adult males. Only one case was seen in 45-60 yrs group.

The largest no. of patients belonged to the VI nerve palsy category. Cases were found in all age groups. The maximal number was in 45-60 yrs age group (10 cases of 23 – 43.47%), followed by 30-45 year group (6 cases of 23 – 26.08%) and > 60 years group (4 cases of 23 – 17.39 %). 6 cases were bilateral cases of VI nerve palsies and one case of bilateral IV nerve palsy .

The category with the least number of patients (only 4 cases – 8%) is the one with multiple nerve involvement. 2 cases were identified in the 45-60 years group (50% - 2 cases of 4), one in each other 30-45 year and > 60 years group.

II - SEX DISTRIBUTION

In the study, there was only a slight gender difference, with males (27 cases of 50 – 54%) outnumbering females (23 cases of 50-46%). In cases with III nerve palsy, the M : F ratio was 5 :3. In cases with IV nerve palsy, the difference was higher with M : F ratio 5 :2. It may be due to the greater outdoor activities and susceptibility to trauma in adult males. Cases with VI nerve palsy of myriad etiologies did not show much gender difference - males (11 cases of 23) and females (12 cases of 23). In contrast, in cases with multiple nerve palsies, females outnumbered males. M : F ratio was 1 : 3.

Table – 2 - GENDER DISTRIBUTION IN CASES OF OCULAR MOTOR NERVE PALSIES

NERVES	MALE	FEMALE	TOTAL OF 50
III	10	6	16
IV	5	2	7
VI	11	12	23
TOTAL	27	23	50

III – LATERALITY :

The difference in laterality was not significant. Right eye was involved in (22 cases – 44 %) whereas the left eye was involved in (21 cases – 42%). There was bilateral involvement in 7 cases. One was a case of IV nerve palsy and the other 6 were cases of VI nerve palsy.

Table – 3 LATERALITY IN OCULAR MOTOR NERVE PALSIES

NERVES	RIGHT	LEFT	BILATERAL
III of 16	10	6	-
IV of 7	4	2	1
VI of 23	6	11	6
MULTIPLE of 4	2	2	-
TOTAL	22	21	7

IV . ETIOLOGICAL ANALYSIS

For the purpose of analysis , the causes of ocular motor nerve palsies were broadly classified into 7 categories

1. Congenital
2. Trauma
3. Vascular(vasculopathic)
4. Neoplastic
5. Aneurysm
6. Miscellaneous / others
7. Idiopathic

Table – 4 : THE VARIOUS ETIOLOGIES OF OCULAR MOTOR NERVE PALSIES

Causes	III (16)	IV (7)	VI (23)	Multiple nerves (4)	Total (50)
Congenital	-	2	-	-	2
Trauma	4	4	3	-	11
Vascular	7	-	9	1	17
Neoplastic	-	-	3	2	5
Others	1	-	2	-	3
Aneurysm	1	-	-	-	1
Un determined	3	1	6	1	11

III – NERVE PALSIES :

The commonest cause of III nerve palsy was vasculopathic (due to diabetes, hypertension and arteriosclerosis in a few cases) i.e.7 cases of 16 – 43.75%. The next in incidence was due to trauma (4 cases of 16-25%). Only one case was due to posterior communicating artery aneurysm.

3 cases were undetermined. The cause could not be identified after routine investigations. The one case in the 'others' category was due to ophthalmolplegic migraine. Of the 4 cases due to trauma, 3 cases were due to head injury and one due to orbital injury. One of them presented with a relative afferent pupillary defect and other with traumatic optic neuropathy.

IV - NERVE PALSIES :

4 cases were due to trauma. The incidence of traumatic nerve palsy was greater in adult males esp. in 15-45 years group. One of them had a bilateral IV nerve palsy. Two of them manifested in the 15-30 year group due to a decompensated congenital IV nerve palsy. One case was idiopathic, even after doing routine investigations. The one case of bilateral IV nerve palsy was due to trauma .

VI NERVE PALSIES :

This group had the myriad variety of causes, in all age groups. 9 cases were due to vascular etiology (39.13%). It was due to diabetes or hypertension or both. Three cases each (13.04 %) were due to trauma and neoplasm. One of them had a acoustic neuroma with VIII nerve and VII nerve involvement.

The other had nasopharyngeal mass lesion. One patient had multiple intracranial space occupying lesions– probably a metastases who expired shortly. 6 cases were undetermined due to a nonspecific neuritis, even after detailed investigations. 2 cases were in the miscellaneous category, which occurred as a post meningitic sequel in pediatric age group.The causes of bilateral VI nerve palsies were diverse .

MULTIPLE CRANIAL NERVE PALSIES :

Only 4 cases were identified. 2 of them were due to neoplasms – one due to pituitary apoplexy from a pituitary tumour, other due to a retro orbital mass. One case was identified in a diabetic and hypertensive. One patient was presumed as Tolosa hunt syndrome.

V - SYMPTOMS :

The commonest complaint was diplopia. This was most annoying patients with VI nerve and IV nerve palsies. Patients with IV nerve palsies were much incapacitated for near work, climbing down staircases etc. some of the them had vertigo and abnormal head posture. In patients with III nerve palsies, drooping of the lid was the most common complaint. Diplopia was present during recovery or when there was only a partial ptosis. Unbearable headache was also complained in patients with VI nerve palsies and III nerve palsies. Even diabetic patients had severe painful nerve palsies.

VI - AGE GROUP DISTRIBUTION OF OCULAR MOTOR NERVE PALSIES IN MALES – 27 CASES

Age Group (in years)	III	IV	VI	Multiple nerves	Total
1 – 15	1	-	1	0	2 (7.40%)
15 – 30	1	2	1	0	4 (14.81%)
30 – 45	2	2	4	1	9 (33.33%)
45 – 60	4	1	4	0	9 (33.33%)
> 60	2	-	1	0	3 (11.11%)

TABLE – 6 : OCULAR MOTOR NERVE PALSIES

AGE GROUP DISTRIBUTION IN FEMALES - 23 CASES

Age Group (in years)	III N	IV N	VI N	Multiple nerves	Total
1 – 15	-	-	1	-	-
15 – 30	1	1	0	-	3 (13.04%)
30 – 45	2	1	2	-	5 (13.04%)
45 – 60	2	-	6	2	10 (43.48%)
> 60	1	-	3	1	5 (21.73%)

In males, the distribution was greater in 30-45 and 45-60 year group. The next in frequency was in 15-30 year group. Whereas in females, most cases were in 45-60 year age group (43.48%). There was a equal but lesser incidence was in 30-45 and > 60 year age group (21.73%).

VII. III Nerve Palsies :

The III nerve palsy may be total or partial. Out of the 16 cases, 8 cases were total and other 8 were partial.

Table – 7

Extent of Involvement in III Nerve palsy

Causes	Total	Partial
Trauma	3	1
Vascular	3	4
Neoplastic	-	-
Others	-	1
Anerusym	1	0
Idiopathic	1	2
Total	8 (50%)	8 (50%)

There may or may not be pupillary involvement in III nerve palsies 13 were having pupillary involvement. Only 3 did not have pupillary manifestation.

Table – 8

PUPILLARY INVOLVEMENT IN III NERVE PALSY

Causes	Pupil involving	Pupil not involving
Trauma	4	-
Vascular	5	2
Neoplastic	-	-
Others	1	-
Anerusym	1	-
Idiopathic	2	-
Total	13 (81.25%)	3 (18.75%)

VIII - ANALYSIS OF VASCULAR CAUSES :

Table – 9

Distribution of Vascular Etiology – Ocular Motor Nerve Palsies

	III	IV	VI	Multiple Nerves
DM	4	-	4	-
HT	-	-	2	-
DM & HT	3	-	3	1

Diabetes and hypertension were the prime cause of vasculopathic or ischemic ocular motor nerve palsies. Of the two, diabetes was more important than hypertension. 7 cases had coexistence of both. All the affected diabetics had poor glycemic control. Blood sugar was above 250 mg % in most of them of the 7 diabetes, 4 of them were diagnosed as diabetes after the nerve palsy. Other 3 although were diagnosed even before, they were on irregular treatment. Regarding hypertensives, all had a diastolic blood pressure above 110 mm Hg. The systolic blood pressure was between 180 to 140 mm Hg. Two of them were found to be hypertensive only after the nerve palsy, others were on irregular or inadequate treatment. A few of these hypertensives also had arteriosclerosis (peripheral vessel wall thickening) 7 of the diabetics and 5 hypertensive had retinopathy changes. Others did not.

IX – THE RECOVERY PATTERN IN OCULAR MOTOR NERVE PALSIES

The recovery pattern of the various nerve palsies was different. The recovery was noted when the patients came for follow up. Follow up was done at 2-3 weekly intervals for 3-4 months. But a few cases who were referred to neurosurgery, neuromedicine and ENT were lost to follow up after 1 or 2 visits. So the recovery could not be documented in those group of patients. Those who had no recovery or only slightly recovery at the end of follow up period were investigated with more tests and another opinion obtained.The recovery was noted in 4 months in most cases, which was almost complete in 6 months. 2 patients with III nerve palsy showed recovery with aberrant regeneration.

Table – 10

THE RECOVERY PATTERN IN OCULAR MOTOR NERVE PALSIES

Recovery	III (of 16)	IV (of 7)	VI (of 23)	Multiple Nerves (of 4)	Total (of 50)
Full Recovery	10 (62.5%)	3 (42.85%)	12 (52.17%)	1 (25%)	26 (52%)
Partial Recovery	3 (18.75%)	2 (28.57%)	5 (21.73%)	2 (50%)	12 (24%)
No Recovery	-	-	3 (13.04%)	1 (25%)	4 (8%)
Lost to follow up / after referal	3 (18.75%)	2 (28.57%)	3 (13.04%)	-	8 (16%)

DISCUSSION

1. Age : In this study 50 cases of ocular motor nerve palsies. The majority were between 15-60 years (80%). In a study of 1000 cases by James, Rush and Younge[23], patients belonged to age group of 2 months – 91 years with 90% patients older than 19 years. In a study by P.A.C. Tiffin et al[2], the age range was from 1-91 years, in a study comprising 165 cases. The widest range was associated with VI nerve palsies as in the present series.

2. Sex : In this study, males comprised (27 cases – 54%), a slight predominance over females (23 cases – 46%). III nerve and IV nerve palsies were higher in males. VI nerve palsies were almost equal in both groups. Females were commonly (75%) identified with multiple nerve palsies than males.

In the study of Rush & Younge[23], the cases included 522 men and 478 females out of 1000. (a slight male preponderance). In the study by PAC Tiffin et al[2], out of 165 patients, 77 were male and 88 were females, with females group outnumbering males especially with VI and III nerve palsies. The numerical data shows that there is not a remarkable gender difference in incidence noticed in any study.

3. Laterality : In the present series, the right eye was affected in 44% and left eye in 42%. Bilaterality was found in 14%. Similarly in the

study by Rush and Younge[23], 451 were right sided,467 left sided and 82 bilateral. In the study by Tiffin et al[2], 67 were right sided, 86 left sided and 12 bilateral. The laterality was not significant in all these groups.

4. Incidence of nerve palsies : In our study of 50 cases, 16 (32%)had III nerve palsies, 7 (14%) had IV nerve palsy, 23 (46%) had VI nerve palsy and 4 (8%) had multiple nerve palsies.

Table : 1 PERCENTAGE OF INCIDENCE OF VARIOUS NERVE PALSIES

Study Nerves	James, Rush & Younge[23]	Tiffin et al[2]	Present series
III	29%	16.96%	32%
IV	17.2 %	21.21 %	14%
VI	41.9%	56.36%	46%
Multiple	11.9 %	5.45 %	8%

As evident in all studies, VI nerve palsy was the commonest and multiple nerve palsy the rarest.

5. Etiologic analysis of various nerve palsies

Causes of III nerve palsy :

Table : 2 COMPARISON OF CAUSES OF III NERVE PALSY

	James, Rush and Younge[23]	Tiffin et al[2]	Present series
Trauma	16.20 %	0	25%
Vascular	20.68 %	46.42%	43.75%
Neoplastic	11.72%	7.14%	0
Others	14.48 %	14.28 %	6.25%
Aneurysm	13.79 %	0	6.25%
Idiopathic	23.10 %	32.14 %	18.75 %

The incidence of vascular causes was the highest, followed by trauma and undetermined causes. Tiffin et al[2] study showed a similar incidence of vascular causes. The incidence of trauma was significant in the study of Rush and Younge[23]. About 20% cases were undetermined.

Causes of IV nerve palsy

Table – 3 COMPARISON OF CAUSES OF IV NERVE PALSY

	James, Rush and Younge[23]	Tiffin et al[2]	Present series
Congenital	-	-	28.57 %
Trauma	31.97%	25.71%	57.14%
Vascular	18.60%	25.71%	-
Neoplastic	4.06%	0	0
Others	7.56 %	2.86%	-
Aneurysm	1.74%	0	-
Idiopathic	36.04%	45.71%	14.52%

About 28.57 % cases were of congenital etiology, presenting in 15-30 years of age group and the majority of the rest of the causes were traumatic. The rest of the causes were undetermined. In the study by Tiffin et al[2] also, the majority of cases occurred in males as in this present series.

Causes of VI nerve palsy :

Table 4 : COMPARISON OF CAUSES OF VI NERVE PALSY

	James, Rush and Younge[23]	Tiffin et al[2]	Present series
Trauma	16.70 %	3.22%	13.04%
Vascular	17.66%	33.33%	39.13%
Neoplastic	14.55%	1.07%	13.04%
Others	17.89%	27.95%	8.69%
Aneurysm	3.57%	1.07%	-
Idiopathic	29.59%	33.33%	26.08%

In the present series, the majority of cases were vascular as in the study by Tiffin et al[2]. A considerable percentage was undetermined, as in other studies also.

Causes of multiple nerve palsies

Table – 5 COMPARISON OF CAUSES OF

MULTIPLE NERVE PALSIES

	James, Rush and Younge[23]	Tiffin et al[2]	Present series
Congenital	-	-	-
Trauma	21.00%	11.11%	-
Vascular	5.04%	-	25%
Neoplastic	34.45%	11.11%	50%
Others	20.16%	55.55%	-
Aneurysm	10.92%	11.11%	-
Idiopathic	8.40%	11.11%	25%

In the present study, the majority of causes (50%) were neoplastic, one case was due to ischemic etiology and another one was undetermined. But the number of cases were different in each study group.

Table – 6 COMPARISON OF NUMBER OF CASES STUDIED

	James, Rush and Younge[23]	Tiffin et al[2]	Present series
III	290	28	16
IV	172	35	7
VI	419	93	23
Multiple	119	9	4
Total	1000	162	50

The slight differences between the different studies may be due to the gross differences in the no. of patients in each study.

6. CHARECTERISTICS OF III NERVE PALSY:

There was a partial involvement in 8 cases (50%) and total involvement in 8 cases (50%), equal in number. The pupil was involved 13 out of 16 (81.25%) cases and spared in 3 out of 16 (18.75%).

In the study by Tiffin et al[2] the pupil was involved in 32.14% (9 out of 28 cases) and spared in 67.86 %

In the study by Tiffin et al[2], there was total involvement in 10 (35.17%) and a partial involvement in 18 cases out of 28 cases (64.28%).

Table : 7 COMPARITIVE STUDY OF EXTENT OF

III NERVE INVOLVEMENT

	Tiffin et al[2]	Present series
Total	35.17%	50%
Partial	64.28 %	50%

7. Recovery pattern of nerve palsies

Table – 8

COMPARISON OF RECOVERY PATTERN-III NERVE PALSY

Recovery pattern	Tiffin et al[2]	Present series
Full Recovery	64.28 %	62.5%
Partial Recovery	10.71%	18.75%
No Recovery or Lost to follow up	25%	18.75 %

The recovery was total in 62.5%, only partial in 18.75%. The results were comparable with study of Tiffin et al[2] and with Rush & Younge[23], the recovery was 48.27%.

Table 9; COMPARISON OF RECOVERY PATTERN – IV NERVE PALSY

	Tiffin et al[2]	Present series
Total Recovery	48.57 %	42.85%
Partial Recovery	22.85 %	28.57 %
No Recovery or Lost to follow up	28.57 %	28.57%

Full recovery was noted in 42.85%, partial recovery in 28.57 % comparable with Tiffin et al[2] study. Study by Rush and Younge[23] showed 53.48 % recovery rate.

Table – 10

COMPARISON OF RECOVERY PATTERN-VI NERVE PALSY

tern	Tiffin et al[2]	Present series
Total Recovery	61%	52.17%
Partial Recovery	26%	21.73%
No Recovery or Lost to follow up	13%	26.08%

The recovery pattern with VI nerve palsy was 52.17 % where it was complete and partial in another 21.73%. Results were comparable with the study by Tiffin et al[2]. With study by Rush and Younge[23], it was 49.64%.

COMPARISON OF RECOVERY PATTERN-MULTIPLE NERVE PALSIES

Recovery was complete in 25%, partial in 50%. With the study by Rush and Younge it was 36.13%. With the study by Tiffin et al it was 22.22%.

SUMMARY

1. Of the 50 cases studied, the age group ranged from 2-72 years. 80% of cases were in the 15-60 years group. IV nerve palsy occurred in 15-45 year group commonly. VI nerve palsies occurred in a wide range.
2. There was a slight preponderance in males (54%) than in females (46%).
3. The right eye was involved in (44%) 22 cases, left eye in (42%) 21 cases and both eyes involved in 7 cases (14%) 6 due to VI nerve palsy and 1 case of IV nerve palsy.
4. Out of 50 cases, 16 (32%) cases were due to III nerve palsy, 7(14%) cases were due to IV nerve palsy and 23 (46%) cases due to VI nerve palsy. Only 4 (8%) cases had multiple nerve involvement. The VI nerve palsy was the commonest, followed by the III nerve and multiple nerve involvement the least common.
5. Of the III nerve palsy – commonest cause (43.75%) was vascular, traumatic was 25% and undetermined causes (18.75%).
6. Of the IV nerve palsies, the commonest cause was trauma (57.14%), followed by congenital (28.57%)
7. Of the VI nerve palsies, there were a variety of causes. The majority was vascular (39.13%). A significant percentage was undetermined

(26.08%). Neoplasms and trauma also made equal contribution (13.04% each)

8. Of the causes of multiple nerve palsies, 50% were neoplastic, 25% vascular and other 25% undetermined.

9. In general, the vascular causes were predominant (34%). 22% were undetermined after investigations 22% were due to trauma. Only IV nerve palsies due to congenital etiology was seen in 2 patients.

10. Half of the III nerve palsies were partial and other half total. Pupillary involvement was seen in 13 cases (81.25%) whereas it was spared in 3 cases (18.75%).

11. Of the vascular causes, diabetes was the commonest cause, followed by hypertension. 8 patients were diabetic, 2 were hypertensive and 7 patients had both. There was a poor control of systemic disease on investigations. Few of them were diagnosed for the first time after the nerve palsy.

12. There was total recovery in 52%, partial recovery in 24%. 24% were either lost to follow up or had no recovery.

13. The recovery rate was higher and more complete in nerve palsies due to vascular causes. The recovery rate due to neoplasms were only partial or nor recovering.

CONCLUSION

The following conclusions were made from the above observations

1. The cranial nerve palsies occur in a wide age group but was more common between 15-60 years
2. Laterality was not significant
3. Unilateral cases were commoner than bilateral cases
4. Gender difference was not significant
5. VI nerve palsy was the commonest, followed by III nerve, next IV nerve and multiple nerve palsy least common.
6. Of all, the vascular etiology was the commonest. Trauma was the next common.
7. Diabetes and hypertension were common associations either alone or together. Poor systemic control of the above was associated with nerve palsies
8. Those with vascular ocular motor nerve palsies belonged to older age group (45-60 years) when compared to those with trauma who presented at an earlier age.
9. Nearly one – fifth of the cases were undetermined.
10. Neuroimaging done in the early period for all indicated cases improved the diagnostic possibilities. Due to the cost of the tests and patient's affordability, it could not be done in a few patients.

MRA and angiography can improve the detection rates, if done at an early period and reduce the number of undetermined cases.

11. Nearly three fourths had complete or partial recovery

12. A careful history, general and complete ophthalmological workup with necessary basic investigations have to be done in all cases. Timely neuroimaging and specialist opinion are necessary although financial problems have restrained and facilities have to be improved.

PROFORMA

Name

Age

Sex

Occupation

Address

Complaints / History of present illness :

1. Double vision

2. Pain

3. Drooping of lids

4. Defective vision – apart from double vision, any blurring or inability to see due to drooping of lids.

5. Deviation of eyeball - right / left eye / alternating, deviation

6. Abnormal head posture

7. Sensation of rotation of self or surroundings (vertigo / Oscillopsia)

H/o Transient obscuration of vision

H/o Deafness

H/o Trauma - to head / eye - whether associated with seizures / loss of consciousness / bleeding from ear / nose.

H/o suggestive of infections - sinusitis

- ear discharge (Otitis),tinnitus/ defective hearing
- pain, redness, swelling (orbital inflammation)
- headache / neck rigidity / vomiting / altered sensorium / convulsions (meningitis)

Fever / loss of weight and appetite

H/o scalp tenderness / headache / jaw claudication

H/o migraine - associated with vomiting, visual aura

H/o suggestive of endocrine problems

- sweating / cold intolerance / h/o use of thyroid related drugs – to r/o hypo or hyper thyroidism
- amenorrhoea, galactorrhoea, infertility, hypogonadism, impotence / sterility
- Obesity / dwarfism / delayed sexual development

H/o contact with pets

H/o chronic use of drugs – OC pills, Vitamin A, Nalidixic acid etc

H/o recent vaccination / exanthematous fevers

H/o envenomation eg. Snake bite

H/o of any numbness / motor weakness over any part of the body

H/o suggestive of other cranial nerve involvement

- Olfaction, color vision, field of vision, dysphagia, dysarthria, diff. in swallowing – nasal regurgitation,

deviation of angle of mouth, inability to close eyelids.

Decreased sensation over face, deviation of tongue.

Personal History :

Diet Smoking Alcoholism

H/o Diabetes, hypertension, tuberculosis, Syphilis, AIDS, Leprosy in the past or present

Past H/o

Any similar episodes, the duration, treatment and recovery.

Treatment History :

H/o of treatment for any systemic medical or surgical illness.

General Examination :

Anaemia	BP
Cyanosis	Pulse
Jaundice	Peripheral pulses
Lymphadenopathy	
Xanthelasma	Neurocutaneous markers
Foot ulcers	

Ocular Examination :

Facial symmetry	-	Ptosis
		Proptosis
		Frontalis overaction
		Deviation of angle of mouth
Head posture	-	Face turn

Head tilt

Chin position

Deviation - ESO / EXO / Hyper / Hypo / tropia

Unilateral / alternating / intermittent

Primary and secondary deviation, Deviation in 9 positions of gaze and on distance and near fixation.

Examination under Slit Lamp : RE LE

5. Lids
6. Conjunction
7. Cornea
8. Iris
9. Pupil - Note on size, anisocoria, reaction to direct, indirect, near reflex, light near dissociation, dilatation lag, observing for synkinetic responses
10. Anterior chamber
11. Lens

Extraocular movements - ductions and versions

Looking in 9 directions with the patients head straight and target moved.

Parks - Bielschowsky 3 step head tilt test (if SO palsy is suspected)

Evaluation of ptosis :

1. Vertical fissure height
2. Margin reflex distance

3. Upper lid crease
4. Levator function
5. Marcus Gunn phenomenon
6. Bell's phenomenon
7. Increased innervation
8. Collier's sign
9. Lid lag

Evaluation of proptosis :

With Hertel's exophthalmometer.

Axial / eccentric, Joffroy's sign.etc.

Visual Field examination

Hess charting

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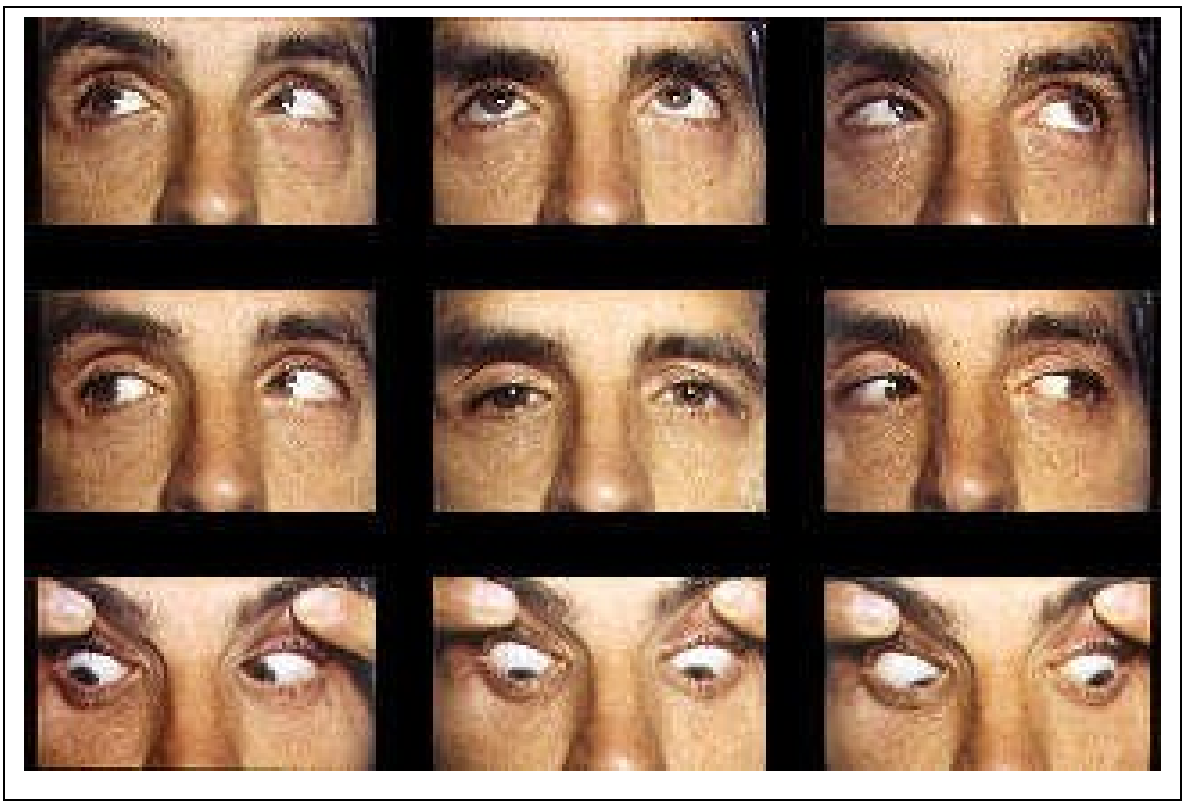
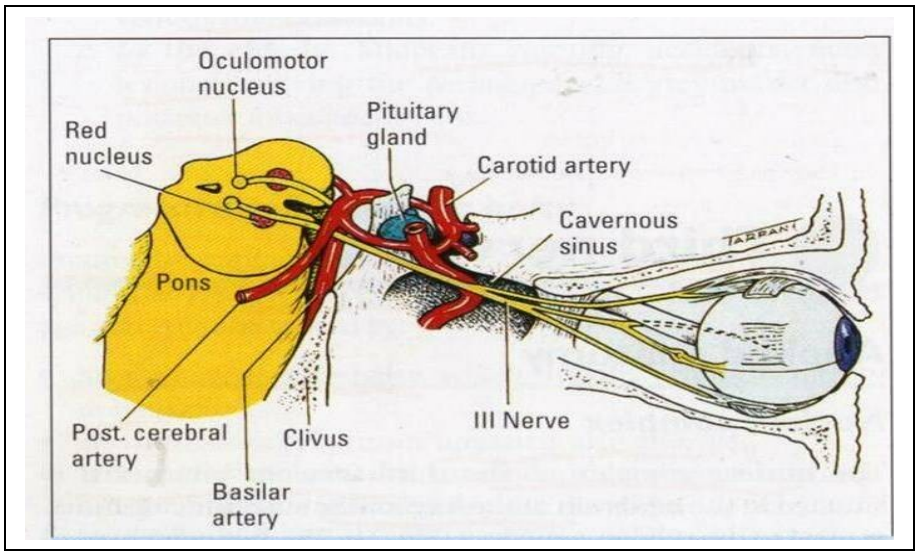
LEFT VI NERVE PALSY

LEFT IV NERVE PALSY

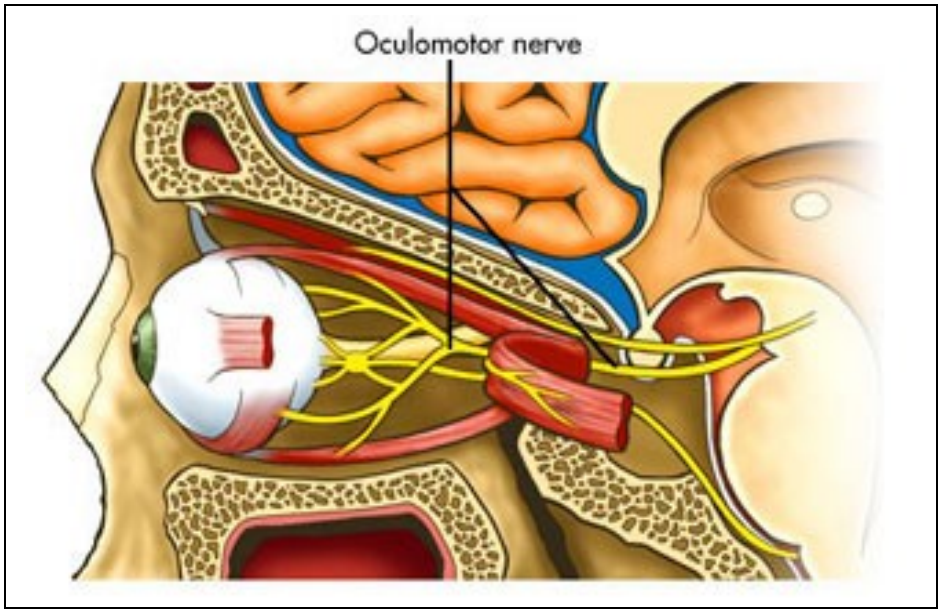


RIGHT III NERVE PALSY

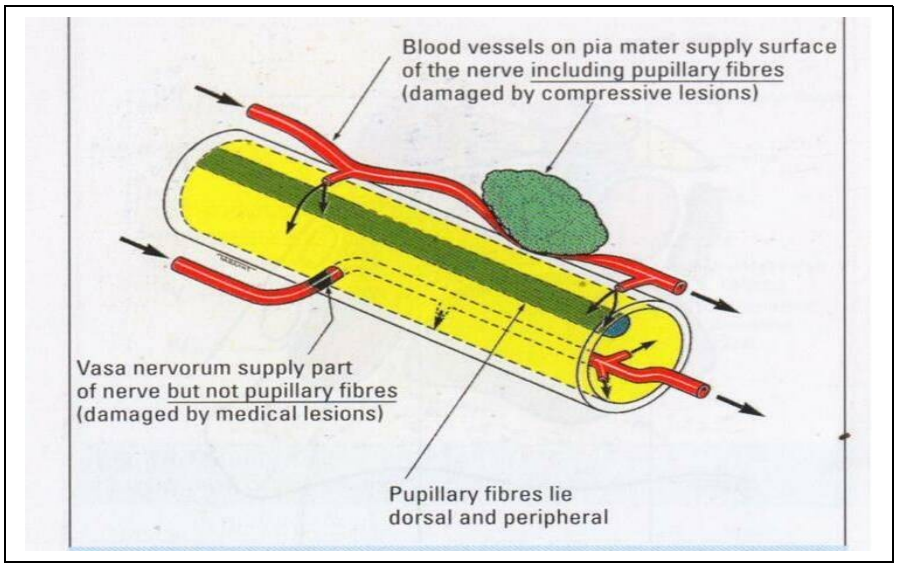
LATERAL VIEW OF THE COURSE OF THE THIRD NERVE



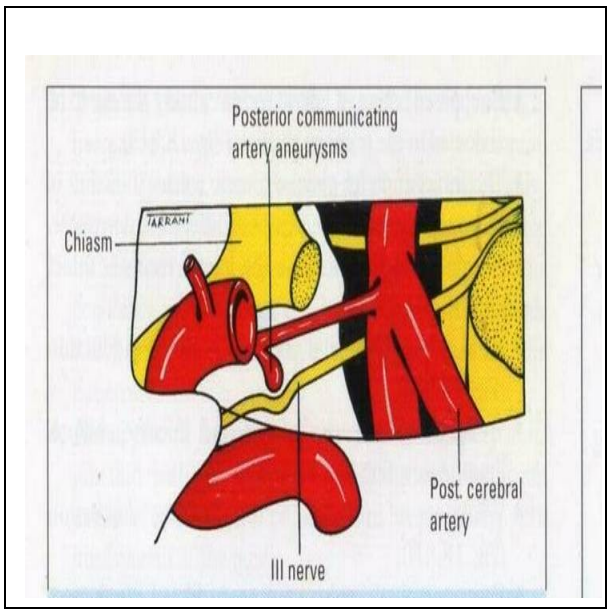
OCULOMOTOR NERVE



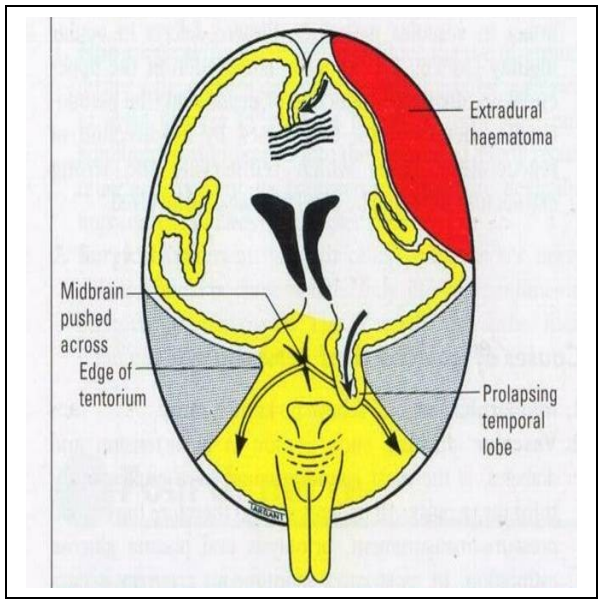
LOCATION OF THE PUPILLOMOTOR FIBRES WITHIN THE TRUNK OF THE THIRD NERVE



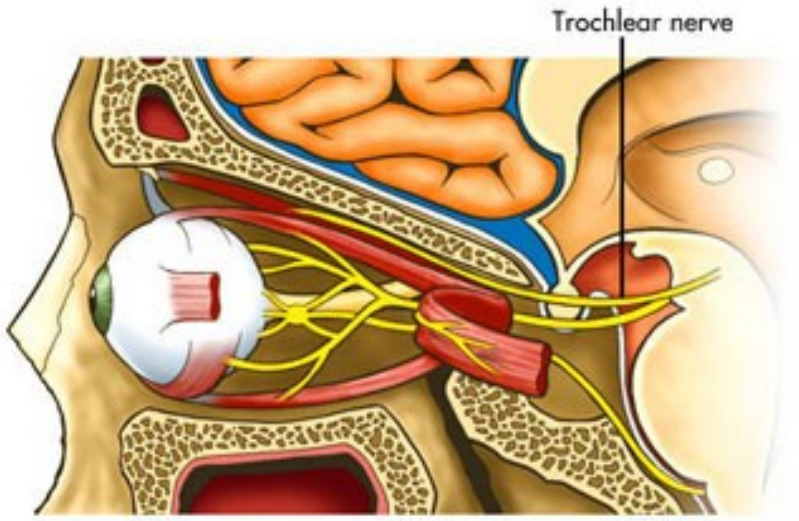
COMPRESSION OF THE THIRDNERVE BY A POSTERIOR COMMUNICATING ANEURYSM



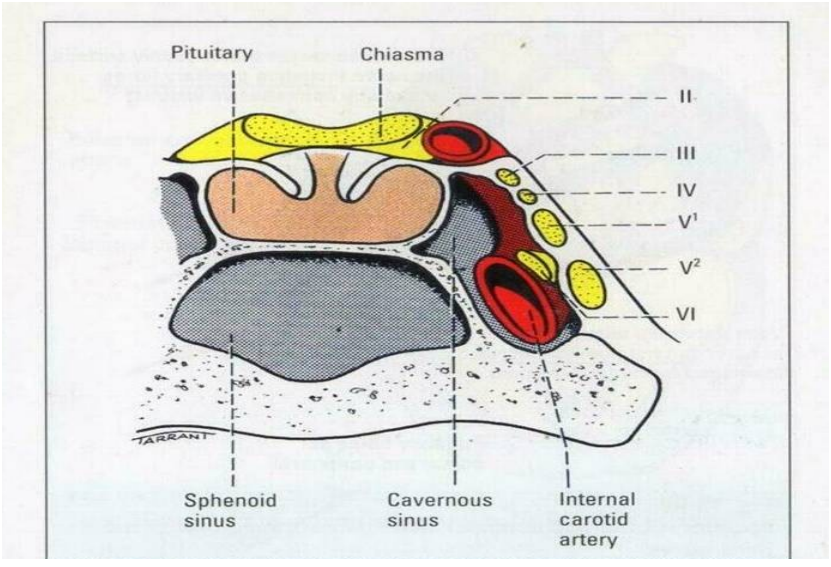
MECHANISM OF THIRD NERVE PALSY



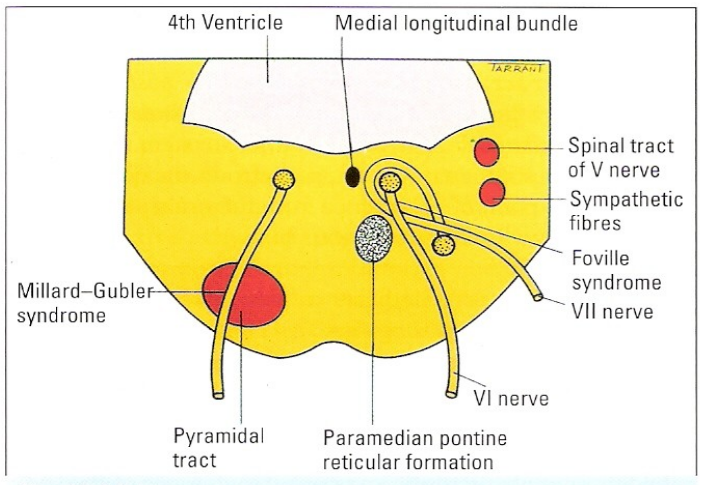
TROCHLEAR NERVE



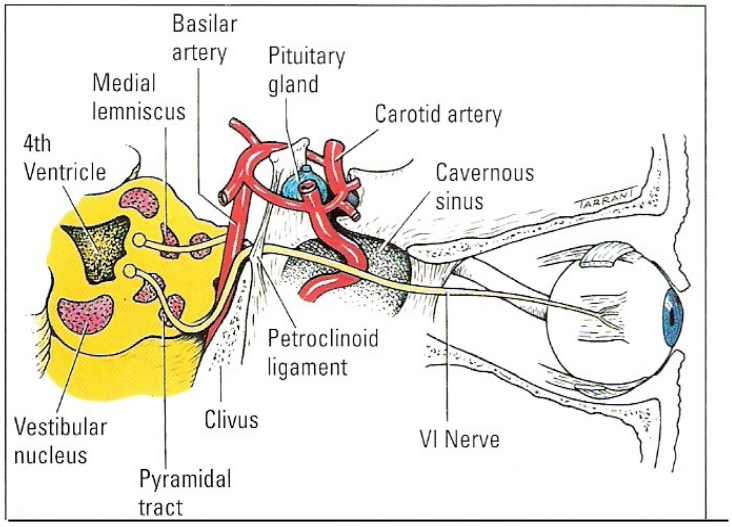
LOCATION OF THE CRANIAL NERVES IN THE CAVERNOUS SINUS VIEWED FROM BEHIND



THE PONS AT THE LEVEL OF THE SIXTH NERVE NUCLEUS



LATERAL VIEW OF THE COURSE OF THE SIXTH NERVE



MASTER CHART

e	Age	Sex	Late rality	Symptoms / complaints	Positive Signs	Positive Investigation Findings	Etiology
	13	M	RE	Drooping of lid headache	Total III N Palsy periorbital edema & echymosis, Head injury	CT Diffuse cerebral edema	Traumati
e	38	F	RE	Unconscious patient	Nerve Palsy Fundus – early papilledema	Brain metastasis (unknown primary)	Neoplasti
i	48	F	RE	Proptosis, defection vision	Retro orbital mass III, IV, VI nerve palsy	CT – optic nerve meningioma	Neoplasti
	20	M	RE	Diplopia – 3 month duration	IV nerve palsy	Previous photos confirm	Congenita
	27	M	RE	Diplopia	VI nerve paresis Fundus –N other cranial N – N	Blood Sugar BP –N CT Brain - N	Idiopathi
van	25	F	RE	Giddiness	IV nerve palsy	MRI – N	Trauma
	18	M	RE	Drooping of lid	Total pupil involving III N palsy with traumatic optic neuropathy	Blood out fracture Orbit R	Trauma
	48	M	LE	Severe pain, headache, drooping of lid	Total pupil involving III nerve palsy	MRA – posterior communicating artery aneurysm	Aneurysm
	62	M	RE	Gidddiness, polyuria, diplopia	VI nerve palsy, Diabetic retinopathy – moderate NPDR BE	Blood sugar FBS 242 mg / dl PP BS 378 mg / dl	Vascular (Diabetes
	66	M	RE	Drooping of lid	Partial pupil sparing III nerve palsy	Blood sugar 302 (on insulin)	Vascular (D
	42	M	LE	Head ache, drooping of lid	III, IV, VI, N palsy Fundus – Normal	CT Normal	?Tolosa Hu syndrome Idiopathi
u	38	M	LE	Diplopia since accident	VI nerve palsy edema, mechanical ptosis, FundusN	CT Brain Normal	Trauma
	32	M	LE	Defective hearing diplopia, deviation of mouth	VI, VII, VIII nerve palsy, papilledema with cerebellar involvement	CT – Left cerebellopontine acoustic neuroma	Neoplasti
	22	F	RE	Giddiness, vertigo (short duration)	IV nerve palsy	N	Congenita
	34	F	BE	Diplopia	IV nerve palsy	-	Trauma
	31	M	LE	Giddiness, Diplopia	VI nerve palsy, fundus – severe NPDR with Gr II HT retinopathy	Blood sugar 309 mg/dl BP 180/110	Vascular (D & HT)
	62	M	RE	Drooping of lid Diplopia	Partial Pupil sparing III nerve palsy partial ptosis	CT Normal	Idiopathi
	14	F	RE	Diplopia, Headache	VI nerve palsy	CT Brain Previous meningitis sequel	Others Po meningiti sequel
n	41	M	LE	Headache, Defective vision	RE – Immature cataract, LE – Mature cataract With LE VI N palsy	Blood sugar – 234 BP - 160 / 100	Vascular (DM & H
	61	F	BE	Headache, vomiting, defective vision	III, IV, V, VI nerve palsy optic atrophy BE	CT – Pituitary apoplexy	Neoplasti
	36	M	RE	Diplopia, abnormal	IV nerve palsy	Park 3 step test positive	Trauma

				head posture		MRI – N	
	42	F	RE	Diplopia after an accident hit by known person	VI nerve palsy Fundus – central serous retinopathy RE	CT – Normal	Trauma
	68	F	RE	Diplopia (VI N paresis BE)	VI Nerve palsy contralateal (R) hemiplegia	CT – large hemorrhage (cerebrovascular accident) with cerebral edema	Trauma
	40	F	RE	Episodic headache with vomiting with visual alua	Total Pupil involving III N Palsy – migraine Recurrent	MRI – N	Others / Miscella ne
	13	M	RE	Diplopia	III nerve palsy-	CT – Not taken	Previous Trauma
	54	F	LE	Diplopia, abnormal head posture	BE, Severe NPDR with CSME, VI nerve palsy	BS – 409 BP – Normal	Vascular (diabetic)
	52	F	LE	Pain, defective vision	III, IV, VI nerve palsy	BP – 150 / 100	Vascular (D & HT)
	30	M	RE	Drooping of lid	Total pupil involving III nerve palsy	-	Idiopathi
	60	F	LE	Headache	VI nerve palsy, physiologic anisoconia, HyperMature cataract -LE	CT Normal	Vascular (D & HT)
	49	F	LE	Diplopia, pain	Partial pupil sparing III N palsy	DM on insulin, RBS 270 mg%	Vascular (D
	47	M	BE	Dysphagia, diplopia, epistaxis	VI, VII, X nerves VI nerve palsy	ENT – Naso pharyngeal carcinoma	Neoplasti
vari	50	F	LE	Drooping of lid	Total pupil involving III N palsy RAPD +	MRI – N	Trauma
ny	52	M	LE	Pain on the left side of face and neck	VI nerve palsy	CT – Postmeningitic sequel	(Post meningiti others miscellaneo
l	71	F	LE	-	VI nerve palsy	N	Idiopathi
	53	M	LE	Diplopia	Partial pupil involving III N palsy	-	Vascular (D & HT)
	47	F	LE	Headache	VI Nerve palsy Grade III HT retinopathy	Chronic renal failure – HT	Vascular(H
	49	M	RE	Diplopia	IV nerve palsy	CT – tuberculoma	Trauma
	50	M	RE	Drooping of lid	Partial pupil involving III N palsy	-	Vascular (D
	50	F	LE	Diplopia	VI nerve palsy BE – ARMD	MRI Brain Normal	Idiopathi
e	63	F	RE	Defective vision	Total pupil involving III N Palsy Premature cataract BE Moderate NPDR	FBS - 306 PPBS 412	Vascular (D & HT)
a	13	M	BE	Pain	VI nerve palsy peri orbital echymosis	X ray Blow out fracture	Trauma
nal	56	F	BE	Headache	VI nerve palsy Go II HT retinopathy	-	Vascular H
	60	M	LE	Defective night vision	Total pupil involving III N palsy C.optic atrophy BE (Retinitis pigmentosa)	-	Vascular (D
mal	42	F	RE	Defective vision	Total pupil involving III N	Diabetic on irregular	(DM & HT

					palsy BE PDR with CSME	treatment	Vascular
al	49	F	LE	-	VI nerve palsy mature cataract RE	MRI – N	Idiopathi
an	42	M	LE	Diplopia vertigo	IV nerve palsy	N	Idiopathi
aj	38	M	RE	Drooping of lid	Total pupil involving III N palsy, BE Toxoplasma choroiditis	-	Trauma
mar	58	M	BE	-	VI nerve palsy No DR Drusen BE	BS F 180 PP 220	Vascular (Diabetic
	44	M	LE	Diplopia Headache	Partial pupil involving III N palsy	MRI – N	Idiopathi
a	64	F	BE	Diplopia	VI nerve palsy	MRI – N	Idiopathic

	GRAVE S OPHTHALMOPATHY	MYASTHENIA	OCULAR MYOPATHY	COMBINED III,IV,VI N PALSY
COURSE	Chronic, rarely acute	Acute / chronic, intermittent	chronic	Acute / chronic
BILATERALITY	usually	Usually , may alternate	always	rarely
PAIN	+ / - foreign body sensation	No	no	variable
PUPILS	normal	normal	normal	variable
TENSILON TEST	negative	positive	negative	negative
FORCED DUCTION	positive	negative	variable	negative
OTHER SIGNS	Lid retraction , scleral injection, proptosis , lid edema, classic echographic , CT or MRI changes	Ptosis , lid fatiguability, orbicularis weakness,Cogan lid twitch sign	Ptosis , orbicularis weakness, + /- temporalis wasting	+ / - trigeminal hyposthesia